DISSERTATION

ON

HYPONATREMIA - A PREDICTOR OF SHORT TERM MORTALITY IN ACUTE ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI)

M.D. DEGREE EXAMINATION

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THANJAVUR

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CHENNAI – TAMILNADU

APRIL 2011

CERTIFICATE

This is to certify that the dissertation entitled "**HYPONATREMIA - A PREDICTOR OF SHORT TERM MORTALITY IN ACUTE ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI) "** is the bonafide work done by **Dr. PREM KRISHNA ANANDAN** in the Department of General Medicine , Thanjavur Medical College , Thanjavur during his Post Graduate Course from 2008 – 2011 . This is submitted as partial fulfilment for the requirement of M.D. Degree Examinations – Branch I (General Medicine) to be held in APRIL 2011.

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DECLARATION

I, **Dr. PREM KRISHNA ANANDAN** solemnly declare that the dissertation entitled "**HYPONATREMIA – A PREDICTOR OF SHORT TERM MORTALITY IN ACUTE ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI) "** is a bonafide work done by me at Thanjavur Medical College Hospital, Thanjavur during September 2009 – October 2010 under the guidance and supervision of **PROF.DR.S.MUTHUKUMARAN.M.D.,** Professor and HOD, Department of Internal Medicine. The dissertation is submitted to **THE TAMILNADU Dr. M .G .R. MEDICAL UNIVERSITY , CHENNAI, TAMILNADU** as partial fulfilment for the requirement of M.D. Degree Examinations – Branch I (General Medicine) to be held in April 2011.

Place: Thanjavur

Date :

Dr. PREM KRISHNA ANANDAN

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INTRODUCTION

INTRODUCTION

Coronary artery disease is the leading cause of death globally[1]. In 2001 coronary artery disease accounted for 7.1 million deaths world wide[2,3], 80% of which were in low income countries like India[4]. It has been estimated that by 2010, 60% of world's heart disease are expected to occur in India[5].Indians are prone to get coronary artery disease at an earlier age than do people in developed countries because of the high prevalence of risk factors like diabetes and hypertension[6,7].

In Indian population ST segment elevation myocardial infarction is the most common type of acute coronary event and contributes to 60.6% of overall incidence of acute coronary syndrome[8].The overall mortality in STEMI is approximately 4 to 7 % or even less in the published clinical trials. However this is not the case in the real world situation[9,10] .This is because the patients enrolled in the randomized trials are selected ones and represented low-risk subgroup. Therefore the results of these trials are not applicable to 50% of patients in clinical practice[11].

A realistic view can be obtained from registry data. In India, CREATE registry data recorded an in-hospital mortality rate of 7.9% and 30 day mortality rate of about 8.6%, which included both patients with unstable angina and AMI. V.Jacob Jose and Satya N. Gupta from Vellore (Tamilnadu), observed 16.9% in hospital mortality rates among South Indian population following STEMI [12].

Hyponatremia is a common electrolyte disorder amongst hospitalized patients[13,14,15,,16],especially with heart failure, nephrotic syndrome or cirrhosis.

Hyponatremia has been shown to be a predictor of cardiovascular mortality among patients with heart failure[17,18,19]. In fact, the neurohormonal activation that accompanies acute myocardial infarction is similar to that which accompanies heart failure[20].

Hyponatremia is common after MI[21], and clinical improvement is accompanied by a rise in plasma sodium concentration[22]. However, while the prognostic value in hyponatremia in chronic heart failure is well established[23,24,25], the prognostic importance of hyponatremia in the setting of acute myocardial infarction are lacking.

This study was done to determine the prognostic importance of hyponatremia in the setting of acute ST elevation MI and to determine its usefulness in predicting short term survival.

AIMS OF THE STUDY

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1. To study the prevalence of hyponatremia in acute ST elevation myocardial infarction.

2. To study the relationship between severity of hyponatremia and short term mortality.

3. To find out the prognostic importance of hyponatremia in acute ST elevation myocardial infarction.

4. To assess the usefulness of hyponatremia as an independent risk factor in predicting short term mortality.

5. To find out the association between hyponatremia and other risk factors like ejection fraction, hypertension, diabetes, smoking, age, sex and type of infarction.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

ACUTE MYOCARDIAL INFARCTION

MAGNITUDE OF THE PROBLEM: [26]

Cardiovascular disease accounts for approximately 12 million deaths annually and to the common cause of death globally. Since past 3 decades there is considerable decline in incidence and prevalence of coronary artery disease in the industrialized western world, whereas it is increasing in the developing world.

Asian Indians, whether living in their own country or as immigrants have much higher incidence of coronary artery disease as compared to all other ethnic groups. Coronary artery disease among Asian Indians has been found to be more severe, diffuse and associated with serious complications and increasing mortality at a younger age.

In this ethnic group specific risk factors, infections and inflammations are emerging. An underlying genetic susceptibility associated with a specific abnormality in lipid profile and different lifestyle factors makes coronary artery disease to assume a malignant course in Asian Indians.

In 1960 CAD represented 4% CVD deaths. In 1990 the proportion was greater than 50% of CVD deaths. CAD deaths rates currently are 3 times more than the stroke rates. However it may suggest metabolic differences in response to the urban life style, higher fat diets and lower levels of activity. Further more the proportion of calories derived from fat, much of which comes from dairy products, is significantly higher in India than in other parts of the developing world.

CORONARY RISK FACTORS FOR ASIAN INDIANS[27]

Asians have different and specific risk factors as compared to western population.

NON MODIFIABLE:

Male age > 35 years

Female age > 45 years

Family history of premature CAD (at age < 55)

MODIFIABLE (NONLIPID)

Hypertension ,Cigarette smoking ,tobacco abuse

Diabetes mellitus / insulin resistance syndromes

Apple obesity or body mass index > 22

Homocysteine > 10 micro mol/lit

High PAI-1

MODIFIABLE (LIPID)

Total cholesterol > 150mg/dl

Triglycerides > 150 mg/dl

LDL cholesterol > 100 mg/dl

APO – a lipoproteins > 100 mg/dl

HDL cholesterol < 40 mg/dl males, < 50 mg/dl females.

MODIFIABLE LIPOPROTEIN RATIOS

TC/HDLc > 4.5

LDLc/HDLc > 3.5

APO A/APO B < 1.2

LIPID TETRAD = (LPa *TG *LDLc) / HDLc > 20,000

PATHOPHYSIOLOGY: [28,29]

Virtually all acute infarcts are caused by thrombosis developing on a ruptured atherosclerotic plaque. Such rupture leads to sudden occlusion of the vessel due to liberation of rbc"s, platelets, macrophages, and subsequently lead to thrombus formation.

CLINICAL PRESENTATION: [30]

Pain is the most common presenting complaint and is described as heavy, squeezing, crushing, stabbing or burning, lasting more than 30 minutes. Typically pain involves central portion of the chest or epigastrium and often radiates to the arms.

PHYSICAL FINDINGS: [30]

Anterior infarction have sympathetic nervous system hyperactivity, tachycardia and hypertension. Patients with inferior wall infarction show evidence of parasympathetic hyperactivity ,bradycardia and hypotension.

Precordium is usually quite and apical impulse may be difficult to palpate. Other physical signs are decreased intensity of S1, paradoxical splitting of S2, S3 or S4 may be heard.

In 1967, Killip[31] proposed a prognostic classification scheme in patients presenting with acute MI.

a) Class-I patients are free of rales and a third heart sound.

b) Class-II patients have rales <50% of lung fields, and may or may not have S3.

c) Class-III patients have rales in >50% of each lung field and frequently have pulmonary edema, S3

d) Class-IV patients are in cardiogenic shock.

INVESTIGATIONS

1) ELECTROCARDIOGRAM:[32]

A) REPOLARISATION (ST-T WAVE) ABNORMALITIES

When acute ischemia is transumural the overall ST vector is usually shifted in the direction of outer (epicardial) layers and ST elevation and sometimes tall positive (hyper acute) T waves are produced over the ischemic zone. Reciprocal ST depressions can appear in leads reflecting the contralateral surface of the heart.

B) QRS CHANGES

Necrosis of sufficient myocardial tissue can lead to decreased R wave amplitude or Q wave in the anterior, lateral or inferior leads as a result of loss of electromotive forces in the infarcted area.

Abnormal Q waves can sometimes be associated with subendocardial infarcts and transmural infarcts can occur without Q waves. Loss of depolarization forces in these regions can reciprocally increase R wave in V1 and V2 rarely without causing diagnostic Q waves in any of the leads.

C) EVOLUTION OF ECG CHANGES :

Ischemic ST elevation and hyper acute T wave occur within hours to days followed by T wave inversion and sometimes Q waves in the same lead distribution. These ischemic changes are often associated with QT prolongation. T wave inversion can resolve after days or weeks or persists indefinitely.

2) LABORATORY FINDINGS

A]SERUM MARKERS OF CARDIAC DAMAGE:

I) CREATINE KINASE MB[33]

CK-MB starts to rise within 4 – 8 hours after the onset of infarction, peaks by 24 hours and declines to normal within 2-3 days.

II) CARDIAC SPECIFIC TROPONINS[33,34,35,36]

Both TnT and TnI first begin to rise above reference limit by 3 hours from the onset of chest pain, TnI peaks by 24 hours and persist for 7 – 10 days after AMI, elevation of TnT peaks by 12 hours to 2 days and may persist up to 14 days.

III) MYOGLOBIN:[,33,34,35]

Peak levels of serum myoglobin are reached considerably earlier (1-4 hours) than peak values of serum CK and its measurement has been suggested as a useful index of successful reperfusion, and even infarct size[33,34].

IV)LACTATE DEHYDROGENASE (LDH):

LDH starts to rise 24-48 hours after the onset of AMI, reaches a peak by 3-6 days and returns to normal levels 8-14 days after the infarction.

V) NEWER CARDIAC MARKERS UNDER DEVELOPMENT [37,38]

1) Heart fatty acid binding proteins (HFABP),

2) Myosin light chains (MLC),Myosin heavy chains (MHC)

3) Glycogen-phosphorylase isoenzyme BB (GPBB) .

B] LIPID PROFILE

Lipid profile should be obtained after 24 – 48 hours[39,40,41] of MI because, for 24 hours total cholesterol and HDL cholesterol remains at or near baseline and generally fall after that.

C] OTHER INVESTIGATIONS

1) LEUCOCYTOSIS[42]

Leukocytosis develops within 2 hours after MI and reaches peak by 2 days ,returns to normal by one week.

2) ESR

ESR is usually normal during 1st or 2 days after MI, rises to peak by 4– 5 days and remains elevated for several weeks.

3) HEMATOCRIT

Hemotocrit often rises following MI due to hemoconcentration.

4) CRP[43,44]

An elevated C-reactive protein (CRP) level appears to identify patients at increased risk of coronary heart disease.

3) IMAGING

A] CHEST X-RAY

The degree of congestion and the size of the left side of the heart on the chest-film are useful for defining groups of patients with MI who are at increased risk of dying after the acute event .

B] 2D ECHOCARDIOGRAPHY

Regional wall motion abnormalities, Ejection fraction estimation helps in establishing prognosis after MI. Echo aid"s in early detection of potentially viable but stunned myocardium , residual provocable ischemia, patients at risk of developing CCF and mechanical complications after MI.

C] DOPPLER ECHOCARDIOGRAPHY

For assessment of blood flow in the cardiac chambers and across cardiac valves. Helps in detecting severity of MR or TR after MI, identification of site of acute ventricular septal rupture, quantification of shunt flow across the defect, and assessment of acute cardiac tamponade.

D] NUCLEAR IMAGING[45]

Radionuclide angiography, perfusion imaging, infarct avid scintigraphy,positron emission tomography have been used to evaluate MI. Cardiac radionuclide imaging for the diagnosis MI is indicated when the triad of clinical history, ECG findings and serum markers are unreliable or unavailable.

COMPLICATIONS OF MYOCARDIAL INFARCTION

1. Left ventricular failure

2. Hypotension

3. Cardiogenic Shock

4. Mechanical Complications, Free wall rupture, Rupture of interventricular septum, Rupture of papillary muscles.

5. Mitral regurgitation

6. Arrhythmias

7 .Electrical instability – Ventricular premature complex,ventricular tachycardia, ventricular fibrillation.

8. Pump failure / excessive sympathetic stimulation .

9.Atrial fibrillation, Paroxysmal supraventricular tachycardia, sinus tachycardia.

10. Bradyarrhythmias and conduction disturbances – Sinus bradycardia AV blocks and Intraventricular Blocks, Junctional escape rhythms.

11. Others: Recurrent chest discomfort, pericardial effusion and pericarditis, Dressler's syndrome,venous thrombosis and pulmonary embolism, Left ventricular aneurysm .

OUTCOME

Outcome in acute MI can be assessed in three phases:

in hospital, early (≤ 30day) and late (beyond 30 days)

MORTALITY:

The overall mortality rate following STEMI is approximately 4-7% in the published clinical trials[9].However, this is not the case in real world situation, which can be obtained from registry data[11].In a study published in Scotland, the case fatality rate is about 22.2%[46]. In MITRA,MIR [47]registry data from Germany, the overall mortality is around 15% .In India, CREATE registry data recorded 30 day mortality rate of about 8.6% in STEMI .Study from Vellore in South India, reported in hospital mortality of 16.9% in a group of 1320 patients with acute STEMI.

MORBIDITY :

Patients with STEMI are having the maximum number of complications because of transmural involvement. Other factors responsible are larger size of infarct and absence of tissue perfusion at the microvacular level[48,49]

HYPONATREMIA

DEFINITION[50]

Hyponatremia is defined as a serum sodium concentration of <135mmol/l after the exclusion of "pseudo-hyponatremia".

ETIOLOGY[51,52,53,54]

CAUSES OF HYPONATREMIA

I. Normal plasma osmolality-

1. Hyperlipidemia

2. Hyperproteinemia

3.Post transurethral resection of prostate

PSEUDOHYPONATREMIA

Extreme elevations in plasma lipids or proteins increase the plasma volume and can reduce the measured plasma sodium concentration.The hyponatremia in this situation does not represent a decrease in extracellular sodium relative to extracellular water .

II. Increased plasma osmolality-

1.Hyperglycemia

2.Mannitol

III. HYPO OSMOLAL HYPONATREMIA

A. PRIMARY SODIUM LOSS (SECONDARY WATER GAIN)

1. Integumentary loss: sweating, burns

2. Gastrointestinal loss: vomiting, tube drainage, fistula, obstruction, diarrhea

3. Renal loss: diuretics, osmotic diuresis, hypoaldosteronism, salt-wasting nephropathy, post obstructive diuresis, nonoliguric acute tubular necrosis.

B. PRIMARY WATER GAIN (SECONDARY SODIUM LOSS)

1. Primary polydipsia

2. Decreased solute intake (e.g., beer potomania)

3. AVP release due to pain, nausea, drugs

4. Syndrome of inappropriate ADH secretion

5. Glucocorticoid deficiency

6. Hypothyroidism

7. Chronic renal insufficiency

C. PRIMARY SODIUM GAIN (EXCEEDED BY SECONDARY WATER GAIN)

1. Heart failure

2. Hepatic cirrhosis

3. Nephrotic syndrome

NEUROHORMONAL ACTIVATION FOR HYPONATREMIA FOLLOWING ACUTE MYOCARDIAL INFARCTION [55]

In acute myocardial infarction, nonosmotic release of vasopressin may occur due to the acute development of left ventricular dysfunction, in response to pain, nausea[56], and major stress, or in response to the administration of analgesics and diuretics[57].

In this setting, vasopressin levels increase concomitantly with the activation of other neurohormones such as rennin and nor epinephrine[58]. However, vasopressin level does not correlate with serum osmolarity in myocardial infarction, suggesting that nonosmotic mechanisms are involved[59].

Activation of carotid barorecepters has been implicated in the nonosmotic release of vasopressin due to arterial underfilling[59].In addition, increased expression of messenger RNA for vasopressin in the hypothalamus has been described.

Moreover, the renal effect of vasopressin is enhanced in heart failure, as the vasopressin-regulated water in the collecting duct is up regulated[60] .

In patients with myocardial infarction, hyponatremia may be aggravated further by the concomitant activation of the renin-angiotensin system and increased catecholamine production[61,62]. These factors decrease the glomerular filtration rate and subsequent delivery of tubular fluid to the diluting segment of the nehpron, further contributing to decreased renal water exertion.

Flear CT, Hilton P[22] in their study of 235 patients admitted to a coronary care unit,concluded that hyponatremia, hypochloraemia, and uremia were common in patients with confirmed myocardial infarctions, the degree of infarctions correlating well with all the above indices of severity. They also found higher in-hospital mortality rates among patients with minimal plasma sodium levels < 130mmol/L.

Szatalowicz VL, Arnold PE, Chaimotivz C, Bichet D, Berl T, Schrier RW in their study have shown that Vasopressin is essential for the development of hyponatremia and arginine vasopressin levels were detectable in 30 of 37 patients with congestive heart failure. They also found that the degree of neurohormonal activation correlates with the severity of hyponatremia in patients with chronic heart failure[63].

Sigurdsson A[20], Held P, Swedberg K in their study of 55 patients with acute myocardial infarction concluded that sustained neurohormonal activation after myocardial infarction mainly occurs in patients with clinical heart failure and is related to the magnitude of myocardial damage, even in patients without heart failure.

Goldberg A et al[64] in their study of 978 patients have concluded that early hyponatremia is a simple marker of neurohormonal activation during the acute phase of myocardial infarction and predicts the long-term development of heart failure and death .

Rouleau JL et al in their study of 534 patients have concluded that neurohormonal activation at the time of hospital discharge in post infarction patients is an independent sign of poor prognosis[65] .

MATERIALS AND METHODS

MATERIALS AND METHODS

50 patients admitted to the intensive coronary care unit of Thanjavur medical college hospital between September 2009 and October 2010, with acute ST elevation myocardial infarction (STEMI) were studied in a prospective manner.

STUDY DESIGN:

1) Single centred
2) Prospective
3) Follow up study

Acute STEMI was diagnosed according to the following criteria.

DIAGNOSIS OF STEMI:

1. Presence of chest pain of >20min duration and

2. ST segment elevation of >1mm in atleast two standard limb leads

or >2mm in atleast two contiguous precordial leads or new onset of

Left bundle Branch block and / or

3. Elevated cardiac biomarkers.

STUDY PARTICIPANTS:

INCLUSION CRITERIA:

Patients who presented within 12 hrs of onset of symptoms, with electrocardiographic evidence of STEMI,elevated cardiac biomarkers and received thrombolytic therapy with streptokinase were included in our study.

EXCLUSION CRITERIA :

1. Patients with Non STEMI or Unstable angina.

2. People with previous history of coronary artery disease.

3. People with previous history of arrhythmias.

4. People with previous history of cardiomyopathy or heart failure.

5.People with previous diuretic use.

6.People with cirrhosis of liver,renal disease,hypothyroidism.

7.Serum Creatinine > 2mg% , Blood urea > 60mg/dl.

Patients who fulfilled the above inclusion criteria and not having any of the above said exclusion criteria were included in the study as a participant.

METHODS

12 lead ECG was taken for all patients, Leads V3R, V4R was taken in patients with inferior wall myocardial infarction.

Location of the infarct were defined as follows

Anteroseptal MI : ST elevation in V1 – V4

Apical MI : ST elevation in V5 – V6

Anterolateral MI : ST elevation in LI,avL,V4-V6

Extensive Anterior wall MI : ST elevation in LI,avL,V1-V6

Inferior wall MI : ST elevation in LII,LIII, aVF

Right ventricular MI : ST elevation in V3R , V4R

Posterior wall MI : Tall and wide R wave, depressed and concave upwards ST, upright and widened T wave in V2.

The data regarding the baseline characters of the patients like age, gender, smoking habit, diabetes mellitus and hypertension were recorded. Hemodynamic status of the patient were assessed by recording the pulse , blood pressure, jugular venous pressure (JVP). Careful auscultation of cardiovascular, respiratory system was done to look for the presence of S3, S4 gallop, murmur and crepitations.All findings were recorded.

Blood sugar, urea, creatinine and electrolytes were done for all the patients at the time of admission. Chest X-ray, lipid profile was taken for all the patients before discharge.

Plasma sodium concentrations were obtained on admission and at 24, 48 and 72 hours thereafter.For every 100mg/dl rise in blood sugar the plasma sodium was reduced by 1.4 meq/dl.

All the patients were treated with antiplatelet drugs , sorbitrate ,morphine, atorvastatin ,ACE inhibitors, betablockers and with accordance to AHA /ACC guidelines as and when required .

All the participants were put on continuous electrocardiographic monitoring for 24 hrs.12 lead ECG was repeated one hour after thrombolysis , every 24 hrs ,and also whenever the situation demanded .

Hemodynamic status was assessed at regular intervals by clinical methods. Arrhythmias were recognized promptly with continuous ECG monitoring and confirmed with 12 lead ECG most of the time. Stable patients were transferred to the medical ward. Ejection Fraction, regional wall motion was analysed with echocardiogram.

After discharge the patients were followed up in the outpatient department weekly for 30 days. Morbidity, mortality data were recorded .

STUDY END POINTS

The primary end point was mortality within 30 days following myocardial infarction. Mortality data after discharge but within 30 days of myocardial infarction were obtained by telephone or postcard returned by patients families or reviewing hospital records.

MEASUREMENT OF SERUM SODIUM

Plasma sodium concentrations were determined by using an ion selective electrode auto analyzer (Roche OMNIC). Hyponatremia was defined as sodium level less than 135mmol/L (<135 mEq/L).

STUDY PROTOCOL

Approval for this study was obtained from Research and ethical committee of Thanjavur Medical College, Thanjavur. Written and informed consent was obtained from all patients. Data were derived from patients by direct interrogation and examination.

STATISTICAL METHOD

All the data were analyzed with SPSS software (version13.0)

Odd"s ratio,Confidence interval,Mean , standard deviation were calculated. Suitable parametric and non parametric tests (Chi square test for non continuous variables,Analysis of variance for continuous variables Z test), Univariate and multivariate logistic regression tests were used to determine the association between hyponatremia and 30-day mortality.

A probability value of <0.05 was considered statistically significant.

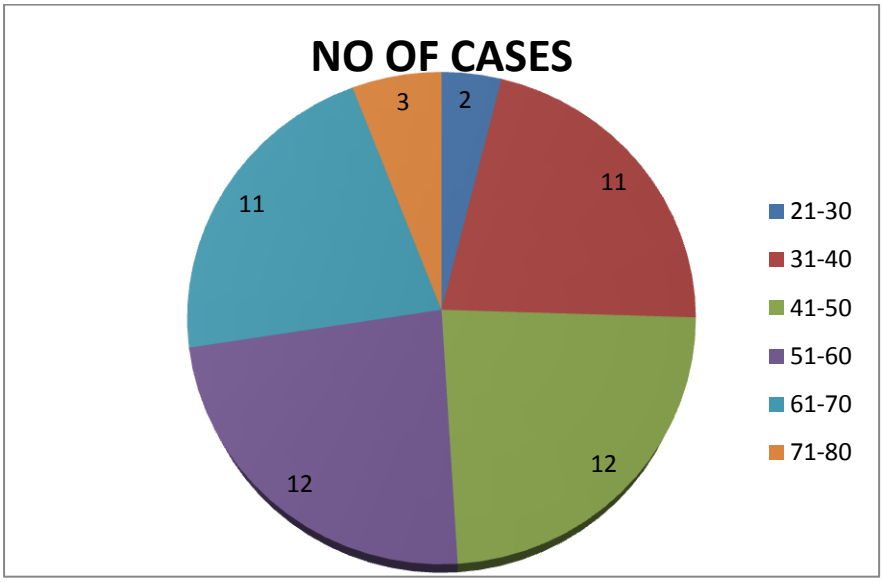
RESULTS

OBSERVATIONS AND RESULTS

TABLE NO 1 –AGE DISTRIBUTION OF CASES

AGE	NO OF CASES	PERCENTAGE
21-30	2	4%
31-40	11	22%
41-50	12	24%
51-60	12	24%
61-70	11	22%
71-80	2	4%

GRAPH 1- AGE DISTRIBUTION



In our study majority of the cases were in the age group of 41-60. Minimun age was 29 ,maximum age was 80. Mean age was 51.46, SD 12.8.

SEX DISTRIBUTION OF CASES

Table no 2 SEX DISTRIBUTION OF CASES

S.no	Content	Males	Females
1	Numbers	40	10
2	Mean Age	51.35±12.8	52±13.4
4	MEAN SODIUM LEVEL	136.8±3.2	135.7±3.7
5	Death	8	3

In our study 80% were males and 20% were females.M:F-4:1.

GRAPH 2 SHOWING SEX DISTRIBUTION OF CASES

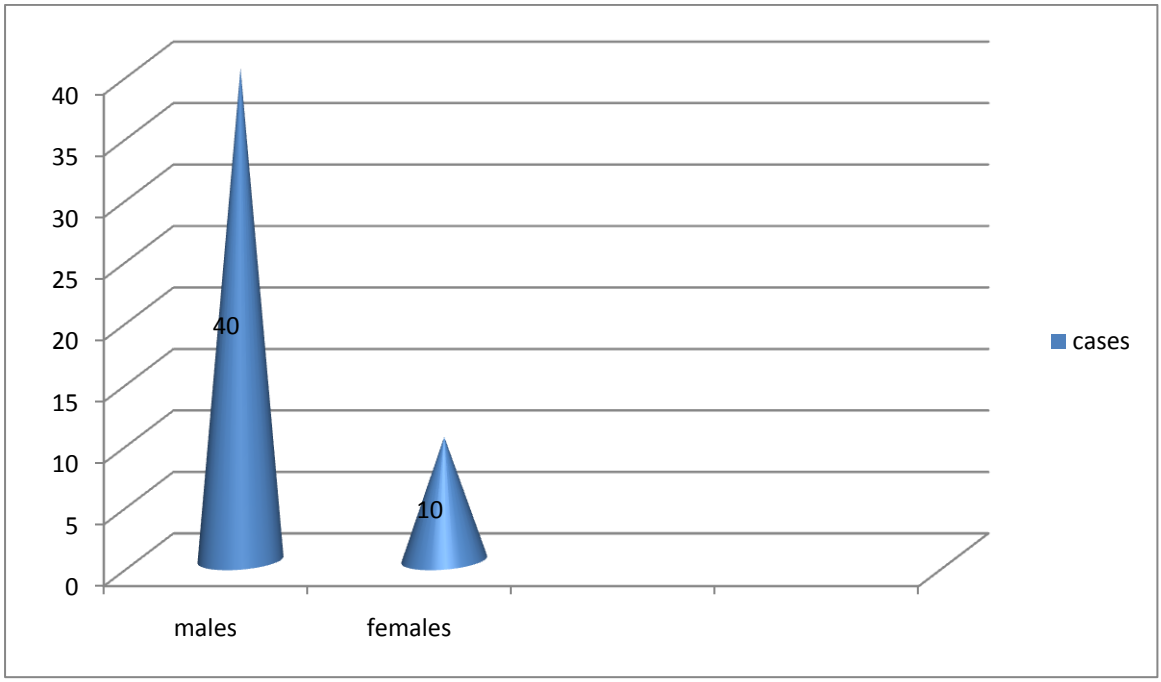
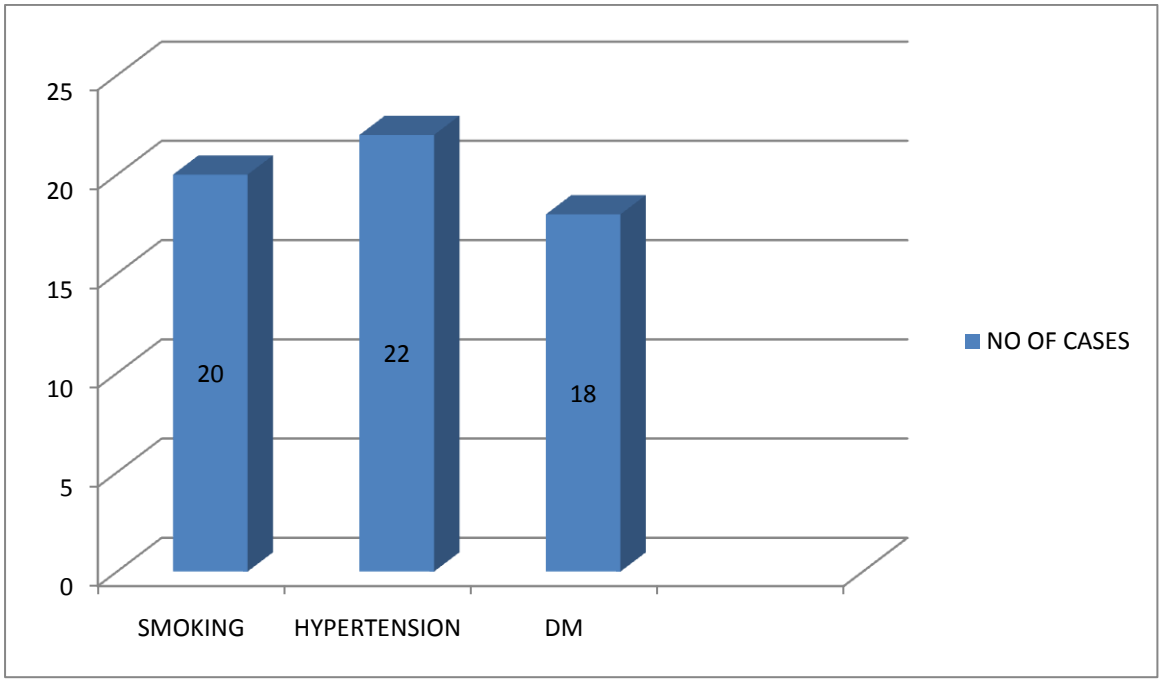


TABLE -3 SHOWING DISTRIBUTION OF RISK FACTORS

S.No.	RISK FACTORS	NO. OF CASES	PERCENTAGE
1	HYPERTENSION	22	44 %
2	SMOKING	20	40 %
3	DM	18	36 %

GRAPH 3 SHOWING DISTRIBUTION OF RISK FACTORS



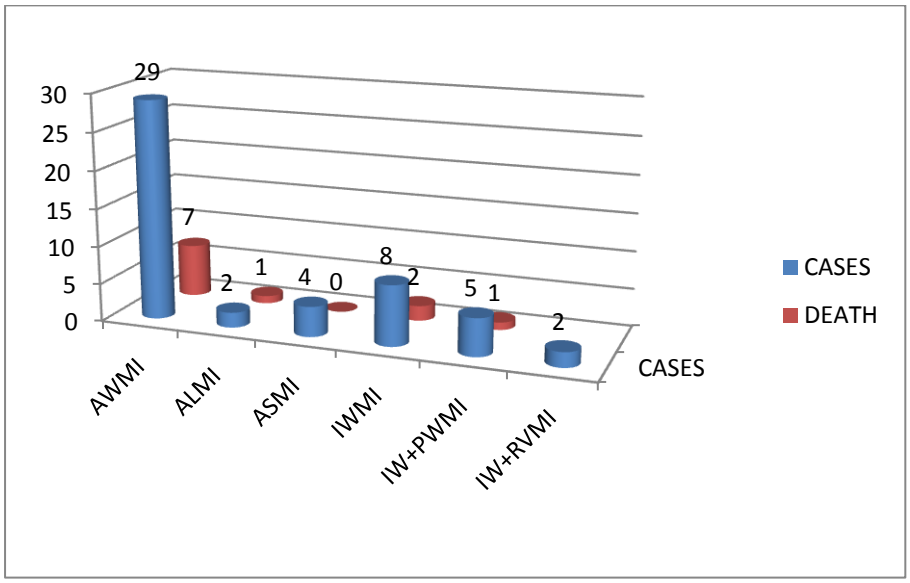
TYPE OF STEMI:

In our study, Anterior wall Myocardial infarction predominated followed closely by inferior wall myocardial infarction.

TABLE-4 SHOWING TYPE OF STEMI:

S.No	Type	No. of Cases	Percentage	Death
1	AWMI	29	58 %	7
2	ASMI	4	8 %	0
3	ALMI	2	4 %	1
4	IWMI	8	16 %	2
5	IWMI+PWMI	5	10 %	1
6	IWMI +RVMI	2	4 %	0

GRAPH- 4 SHOWING DISTRIBUTION OF TYPE OF STEMI



OUR STUDY PATIENTS WERE DIVIDED INTO THREE GROUPS

Group A = patients with normal sodium levels

Group B = patients with hyponatremia on admission

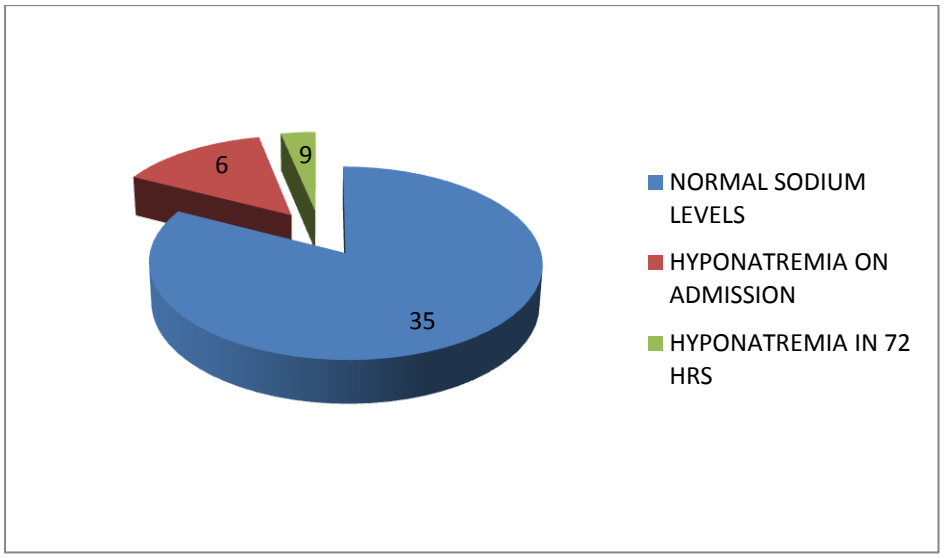
Group C = patients who developed hyponatremia within 72 hours

All base line characteristics of the three groups were compared.

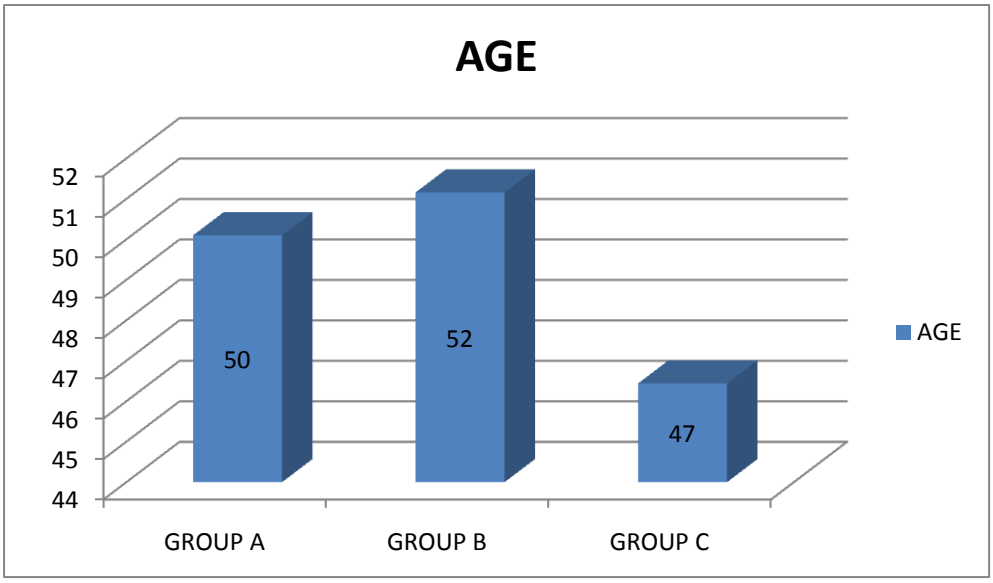
TABLE 5

GROUP	NO.OF CASES
A	35
B	6
C	9

GRAPH 5 SHOWING NO.OF CASES IN EACH GROUP

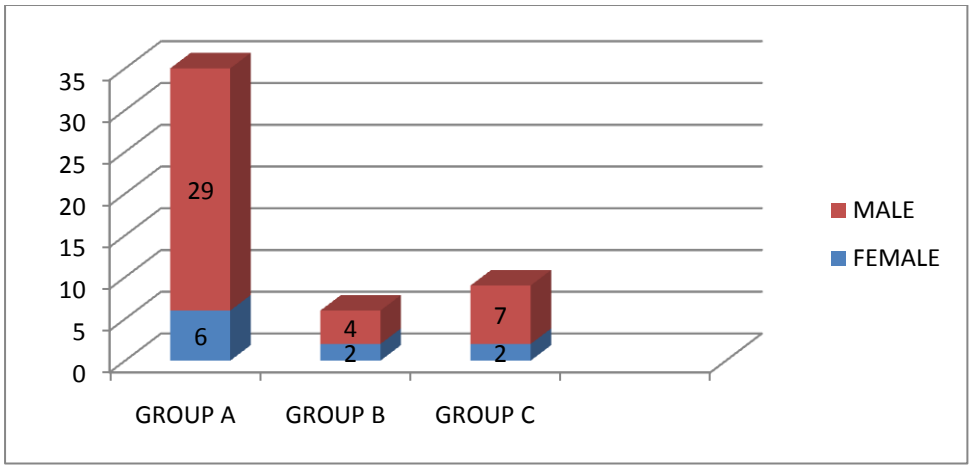


GRAPH 6-MEAN AGE IN STUDY GROUPS



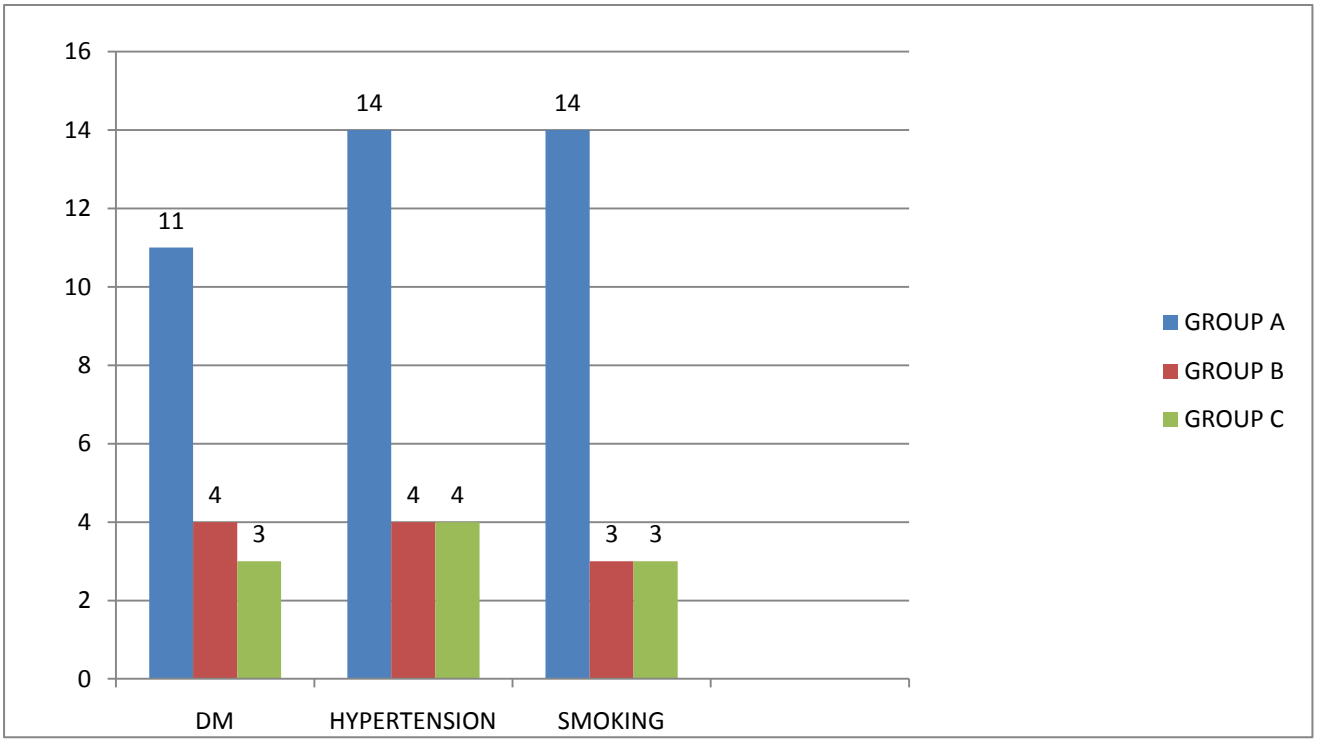
Patients who presented with hyponatremia on admission (52±14) were of higher age when compared to patients with normal sodium levels (50 ±12).

GRAPH 7 SEXWISE DISTRIBUTION IN STUDY GROUPS



As evidenced males constituted majority of the cases in all the three groups.There were 29 males in group A, 4 males in group B and 7 males in group C.There were 6 females in group A, 2 in group B and 2 in group C.

GRAPH 8-RISK FACTORS AMONG THE THREE GROUPS



67% were diabetic , 50% were smokers, 67% were hypertensives among patients who presented with hyponatremia on admission.

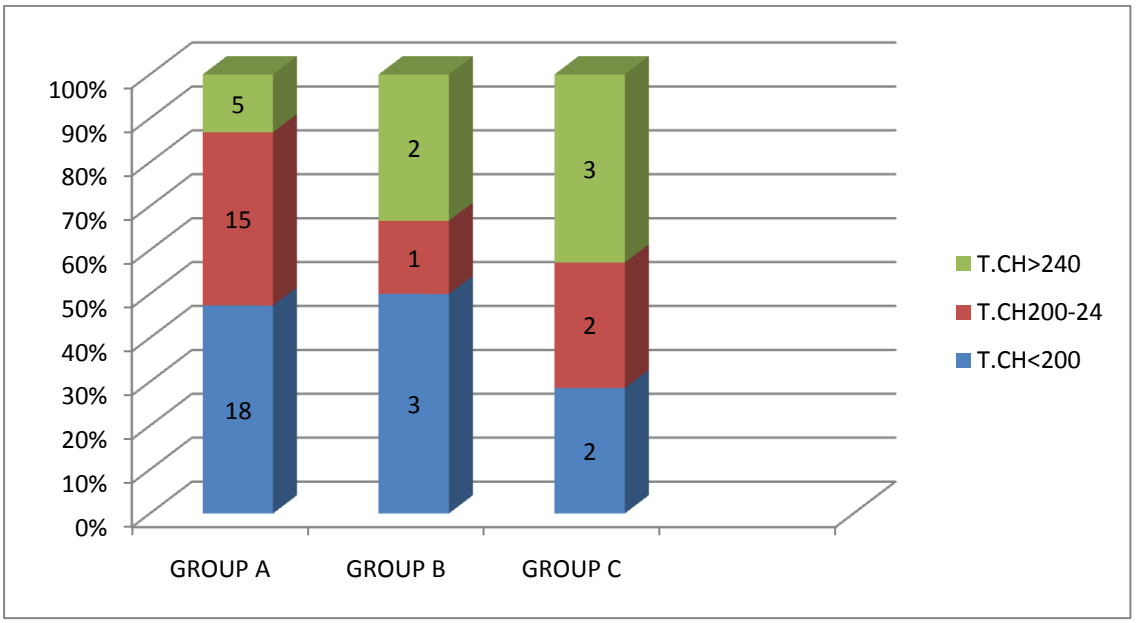
Among patients who had normal sodium levels only 31% were diabetic, 40% were smokers and 40% were hypertensive. The proportion of diabetic, hypertensive and smokers were more among patients with hyponatremia.

TABLE-6 TOTAL CHOLESTROL IN THREE GROUPS

S. NO	CONTENTS	T.CH < 200	T.CH 200 – 240	T.CH > 240
1	TOTAL CASES	23	17	10
2	PERCENTAGE	46%	34%	20%
3	GROUP A	18	15	5
4	GROUP B	3	1	2
5	GROUP C	2	2	3
6	DEATH	4	3	4

The mean cholesterol was found to be 209.18mg/dl and S.D was 29.3

GRAPH 9 -TOTAL CHOLESTROL IN THREE GROUPS

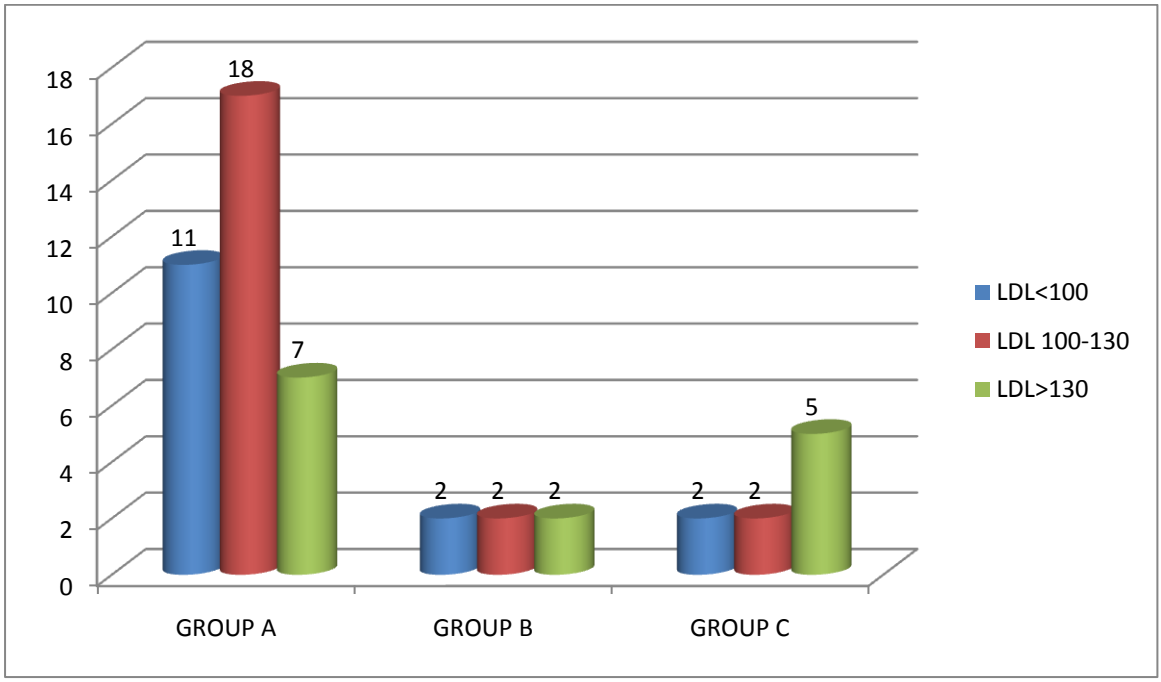


Among patients who had total cholesterol >200 mg/dl 8 patients had hyponatremia.Only 5 patients in the hyponatremia group had total cholesterol <200mg/dl.

TABLE 7: LDL CHOLESTEROL LEVELS IN THREE GROUPS

S.NO	CONTENT	LDL < 100	LDL 100- 130	LDL >130
1	total cases	14	22	14
2	percentage	28%	44%	28%
3	GROUP A	11	18	7
4	GROUP B	2	2	2
5	GROUP C	2	2	5
6	Death	3	4	4

GRAPH 10: LDL LEVELS AMONG THREE GROUPS

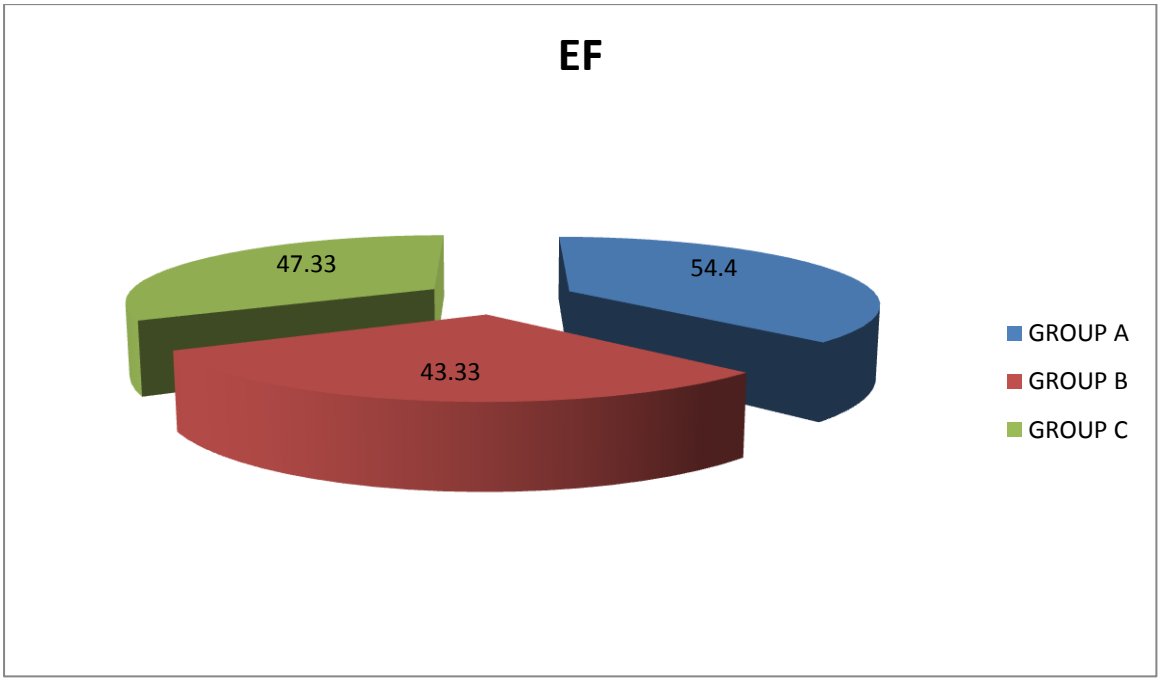


The mean LDL cholesterol was 115.3mg/dl. Levels > 130mg/dl were present in 28%. Among them 7 had normal sodium levels,2 had hyponatremia on admission and 5 developed hyponatremia within 72 hours.

TABLE 8 : EJECTION FRACTIONS AMONG THREE GROUPS

GROUP	MEAN EF
A	54.4 %
B	43.33 %
C	47.33 %

GRAPH 11: EJECTION FRACTION AMONG THREE GROUPS



The mean ejection fraction was lower among patients who presented with hyponatremia or developed hyponatremia within 72 hours when compared with patients with normal sodium levels.

TABLE 9:KILLIP CLASS AMONG THREE GROUPS

KILLIP CLASS	Normal sodium levels(n=35)	Hyponatremia on admission (N=6)	Hyponatremia within 72 hrs (N=9)	DEATH
CLASS I	28 (80 %)	4 (66%)	7 (77%)	6 (54%)
CLASS II	6 (17%)	2 (33%)	1 (11%)	4 (36%)
CLASS III	1 (2%)	0	1 (11%)	1 (9%)
CLASS IV	0	0	0	0

A total of 6 patients with killip class I expired. Among them 4 had hyponatremia on admission and 2 developed hyponatremia within 72 hours and no deaths occurred in patients with normal sodium levels.4 patients with killip class II expired.Among them 2 patients had hyponatremia on admission , 1 patient developed hyponatremia within 72 hours.

GRAPH 12-KILLIP CLASS AMONG THREE GROUPS

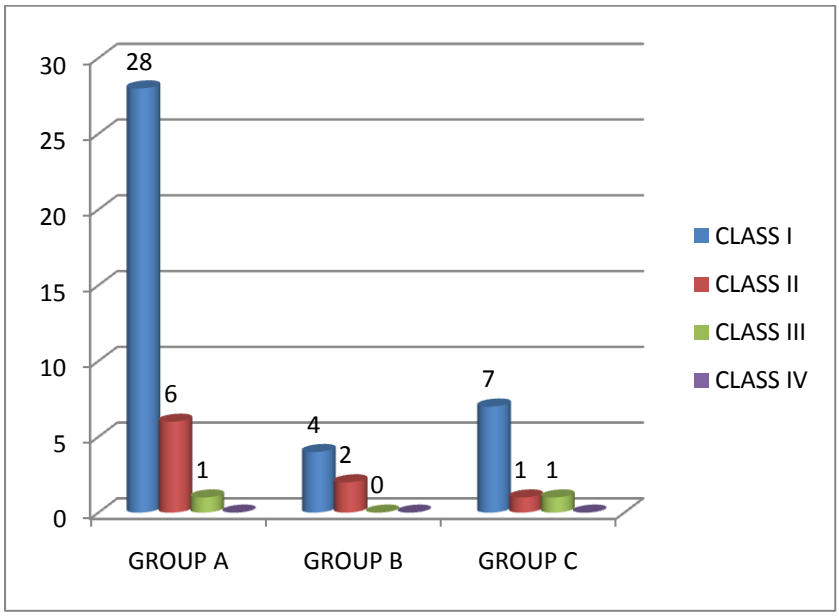


TABLE 10

IN HOSPITAL COMPLICATIONS

S. No	Complication	Count	Percentage	Death	Mean Sodium Level Meq/l
1	CCF	2	4%	1	133.75
2	Acute MR	1	2%	1	129
3	Pulmonary edema	2	4%	1	137.8
5	Heart block	2	4%	0	138.25
6	Ventricular Tachycardia	1	2%	0	134.25

GRAPH 13-IN HOSPITAL COMPLICATIONS

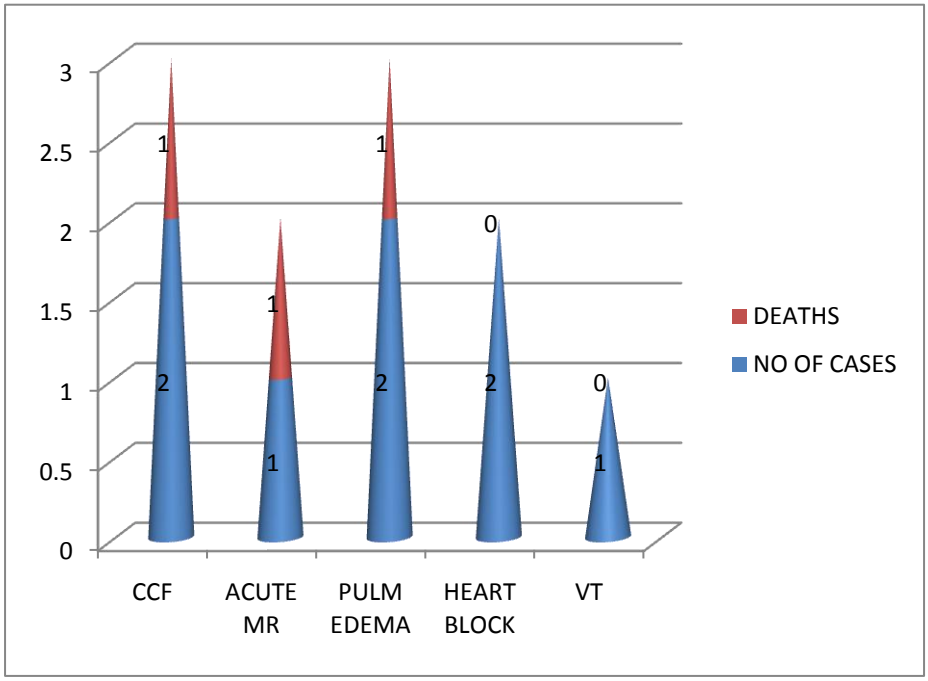


TABLE 11- SHOWING MORTALITY AMONG THREE GROUPS

	NORMAL SODIUM LEVELS	HYPONATREMIA ON ADMISSION	HYPONATREMIA WITHIN 72 HRS	TOTAL
NO. OF PATIENTS	35	6	9	50
MORTALITY	4	5	2	11

In our study, 35 patients had normal sodium levels,6 had hyponatremia on admission and 9 developed hyponatremia within 72 hours.The overall mortality rate was 22% (11 out of 50).

GRAPH 14 - SHOWING MORTALITY AMONG THREE GROUPS

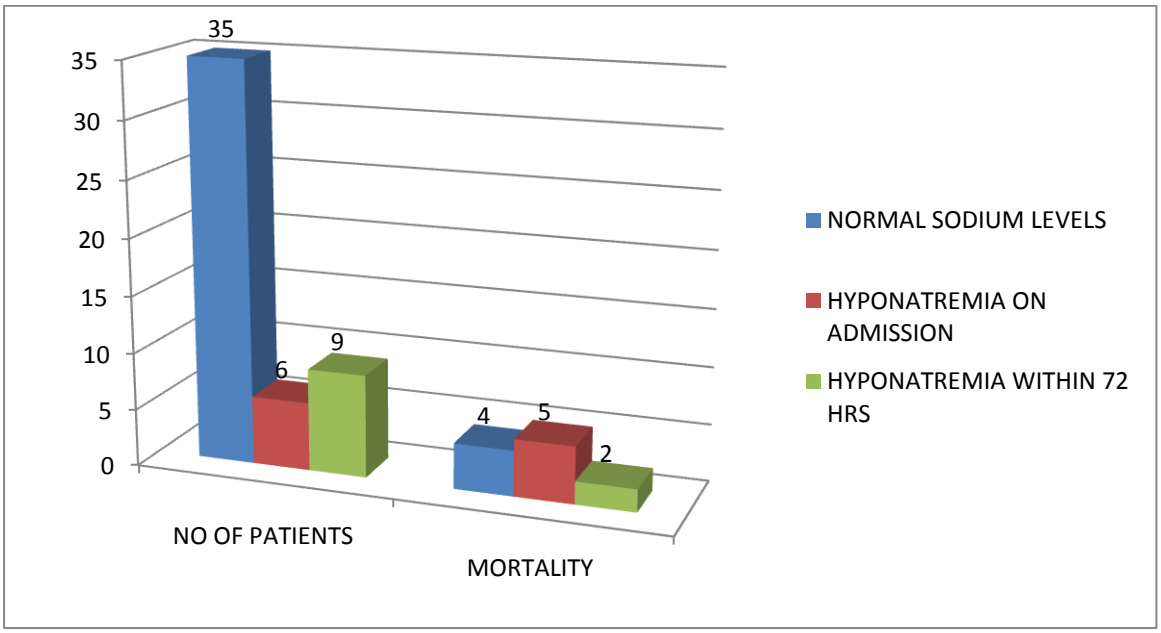


TABLE 12 COMPARING MORTALITY RATES AMONG THE THREE GROUPS

GROUP	MORTALITY RATES
GROUP A	11.4%
GROUP B	83.4%
GROUP C	22%

GRAPH 15 COMPARING MORTALITY RATES AMONG THE THREE GROUPS

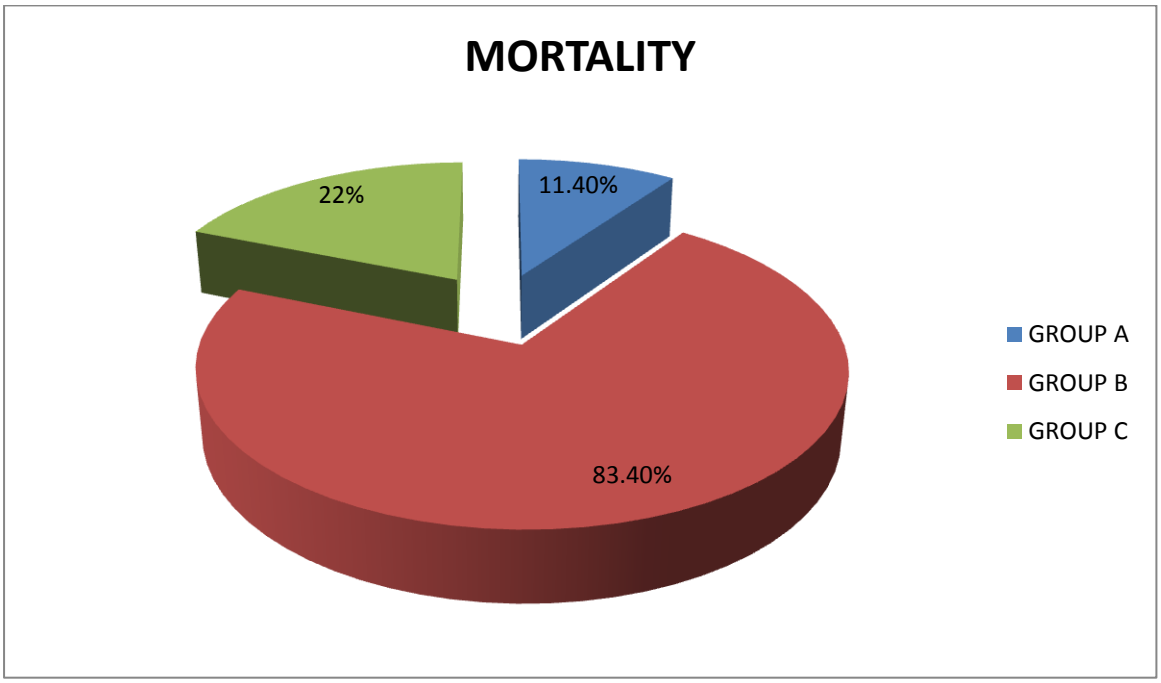


TABLE 13 SHOWING SEVERITY OF HYPONATREMIA AND OUTCOME IN TERMS OF MORTALITY

SODIUM LEVELS	NO.OF CASES	MORTALITY
<130 MEQ/L	4	4(100%)
131-134 MEQ/L	11	3(27.3%)

Among 4 patients with sodium level <130meq/l the mortality rate was 100%. 11 patients had sodium levels between 131-134,out of which only 3 expired.

GRAPH 16- SHOWING SEVERITY OF HYPONATREMIA AND OUTCOME IN TERMS OF MORTALITY

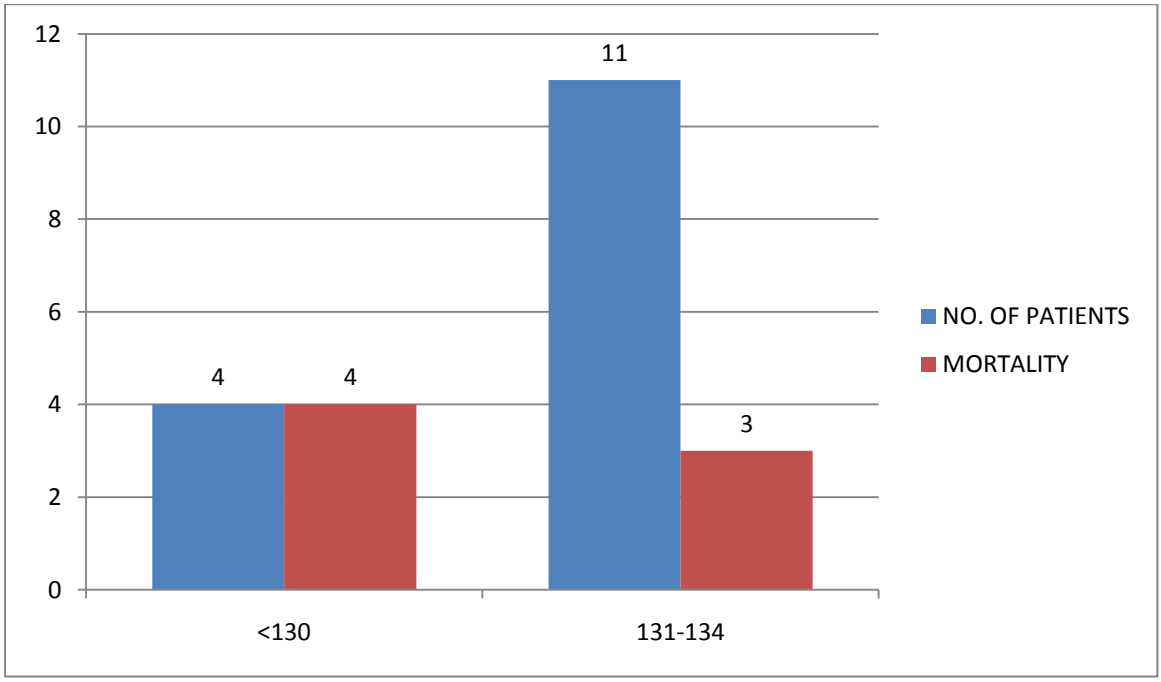


Table 14 SHOWING IN HOSPITAL AND POST DISCHARGE DEATH.

S.NO	NO .OF DAYS	NO. OF DEATHS	MEAN SODIUM
1	<7 days	3	133.3 meq/l
2	8-30 days	8	134.4 meq/l

During the hospital stay three patients expired.Each group had one mortality.During follow up period three patients were re-admitted with post infarction failure and expired. Each group had one mortality. Out of the remaining 5 deaths ,2 patients belonged to group A and 3 belonged to group B. There was no mortality in group C .

TABLE 15 SHOWING CASES READMITTED WITH POST INFARCTION FAILURE AND DEATH.

GROUP	NO. OF CASES	MEAN SODIUM
Group A	1	141
Group B	1	128.75
Group C	1	133

As evidenced from the above table the mean sodium levels were low for patients who got re-admitted with post infarction failure**.**

TABLE16-SHOWING BASE LINE CHARACTERISTICS OF 50 PATIENTS

S.NO	Characteristics	Normal sodium levels(n=35)	Hyponatremia on admission (N=6)	Hyponatremia within 72 hrs (N=9)	P value
1	Age(yrs)	50 ± 12	52 ± 14	47 ± 17	P=0.06
2	Male sex	29(82%)	4(67%)	7(78%)	P=0.04
3	Diabetes	11(31%)	4(67%)	3(33%)	P=0.03
4	Smoking	14(40%)	3(50%)	3(33%)	P=0.05
5	Hypertension	14(40%)	4(67%)	4(44%)	P=0.1
6	Anterior wall infarction	23(66%)	4(67%)	8(89%)	P=0.02
7	T.Cholestrol	208.7±29.78	212±30.1	214±33.4	P=0.6
8	S.ldl	114.5±20.8	121.5±31.3	122.4±29.4	P=0.3
9	Killip class	class I-28 classII-6 class III-1	class I-4 class II-2 class III-0	class I-7 class II-1 class III-1	P=0.001
10	Ejection fraction(%)	54.4±10.7	43.33±7.68	47.33±15.57	P=0.001

All the baseline characteristics among the three groups of patients were compared.

Patients who presented with hyponatremia on admission belonged to a higher age group than patients with normal sodium levels.

67% of patients who presented with hyponatremia on admission and 78% of patients who developed hyponatremia within 72hrs were males.

Patients who presented with hyponatremia or developed hyponatremia within 72 hours more often were smokers(67%) and had diabetes(67%), anterior infarction(67and89%), lower ejection fraction(43.33±7.68) when compared to patients with normal sodium levels.

ODDS RATIO FOR 30 DAY MORTALITY

TABLE NO 17- ODDS RATIO- GROUP A VERSUS OTHER GROUPS

	SURVIVORS	NON SURVIVORS	ODDS RATIO	Z SCORE	P VALUE
GROUP A	31	4			
GROUP B	1	5	38.75	3.004	0.027
GROUP C	7	2	0.057	2.108	0.035

Odds ratio for 30 day mortality was found to be high in hyponatremic Group s (P=0.027 and 0.035) which is statiscically significant.

TABLE NO 18 COMPARING SURVIVORS AND NON SURVIVORS

S.NO	Risk factors	Survivors	Nonsurvivors	T or X^2	P value
	N	39	11		
1	Age(yrs) (mean ±SD)	48.89±11.6	55.1±17.9	1.382	0.173
2	Sex M F	32(82%) 7(18%)	8(72%) 3(27%)	0.7	0.09
3	Hyponatremia (mean ±SD)	135.02±1.65	130.9±2.7	6.29	0.001
4	Smoking	12(61%)	8(72%)	0.09	0.7
5	Diabetes	11(33%)	7(45%)	0.14	0.70
6	Hypertension	14(36%)	7(63%)	1.5	0.2
7	Infarct site Anterior Inferior	 17(43%) 12(31%)	 8(72%) 3(27%)	 1.8 0.01	 0.17 0.9
8	T.Cholestrol	207.38±28.7	215.54±33.21	0.540	0.5
9	S.LDL	113.28±20.8	122.5±25.8	0.53	0.3
10	Killip class I II III	33(85%) 5(13%) 1(2%)	6(54%) 4(36%) 1(9%)	0.62	0.04
11	EF(%)(mean ±SD)	54.35±11.2	42.72±9.2	3.15	0.002

It was seen that serum sodium levels was statistically significant in determining mortality. Mean serum sodium level was 135.02±1.65 in the survivors and 130.9±2.7 in non survivors.

Other factors such as Killip class, ejection fraction, hypertension, diabetes, age , sex,dyslipidemia were found to influence mortality.

Multivariate analysis and analysis of variance was performed which showed that along with other risk factors, hyponatremia on admission ($p<0.001$) or early development of hyponatremia ($p<0.008$) is an independent risk factor in predicting short term mortality.

TABLE NO 19 SUMMARY OF RISK FACTORS IN PREDICTING MORTALITY

VARIABLE	P VALUE
AGE	0.1
SEX	0.09
SMOKING	0.7
HYPERTENSION	0.7
DIABETES	0.2
DYSLIPIDEMIA	0.3
KILLIP CLASS	0.04
HYPONATREMIA ON ADMISSION	0.001
HYPONATREMIA WITHIN 72 HOURS	0.008
EF	0.002

As evidenced from the above table killip class,ejection fraction and hyponatremia on admission or early development of hyponatremia significantly influenced mortality.Multivariate analysis showed that along with other risk factors, hyponatremia was a significant independent predictor of 30 day mortality.

DISCUSSION

DISCUSSION

In acute myocardial infarction the development of hyponatremia is a marker that probably incorporates different prognostic entities,including severe left ventricular dysfunction,hemodynamic alterations,and the extent of neurohormonal activation.

Goldberg A[66] et al studied 1047 patients with acute ST elevation MI, without past history of heart failure.It was found that hyponatremia on admission or early development of hyponatremia was independently associated with short term mortality.

AGE DISTRIBUTION IN MYOCARDIAL INFARCTION

Our study comprised of 50 pateints with acute ST elevation MI. The mean age was 51.4 ± 12.8 .Majority of the cases were in the age group of 41-60 . In the study conducted by Aziz M et al[67] , the mean age was 57.28±6. In Goldberg"s study the mean age was 61±12.When compared to the other studies it is seen that Indians are prone to get MI at an earlier age.

SEX DISTRIBUTION IN MYOCARDIAL INFARCTION

In our study, 80% were males and 20% were females.Simillar results were seen in a studies conducted by Aziz M et al, Goldberg A[66] et al. Thus males are more prone to develop MI.

RISK FACTORS IN MYOCARDIAL INFARCTION

In our study 20 patients were smokers, 22 were hypertensive and 18 were diabetic.Among 11nonsurvivors,72% were smokers,63% were hypertensive,45% were diabetic.Killip and Norris et al [68]in the Framingham heart study said that diabetes and smoking increases the risk of death after myocardial infarction.In GISS-2 trial[69], out of 11483 hypertensive MI patients,3306 patients expired. Our study reveals that Diabetes,Hypertension and smoking are important risk factors in determining mortality.

PREVALANCE OF HYPONATREMIA

In our study, hyponatremia was present on admission in 6 patients (12%). Hyponatremia developed in 9 patients(18%) during the first 72 hours of hospitalisation. In a study conducted by Goldberg A et al,hyponatremia was present in 131 patients (12.5%) and hyponatremia developed in 208(19.9%) during the first 72 hours of hospitalisation.Similar results were seen in the studies conducted by Flear CT et al ,Aziz M et al and Sushrat W et al[70]. Our results were also consistant with other studies.

ASSOCIATION BETWEEN HYPONATREMIA AND AGE

Patients who presented with hyponatremia on admission belonged to a higher age (52 ± 14) when compared to patients with normal sodium levels who belonged to younger age (50 ±12). In the study conducted by Goldberg A, Hammerman H et al, mean age among patients with normal sodium levels was 61±13 and hypontremic individuals was 63±13.

ASSOCIATION BETWEEN HYPONATREMIA AND SEX

Males constituted majority of the cases .There were 29 males(82%) with normal sodium levels,4 males(67%) with hyponatremia on admission and 7 males(78%) developed hyponatremia within 72 hours after admission.There were 6 females with normal sodium levels ,2 with hyponatremia on admission and 2 developed hyponatremia within 72 hours after admission.The higher male ratio was due to the fact that number of females were less in our study.Simillar results were found in Goldberg A , Hammerman H et al,Aziz M et al “s study.

ASSOCIATION BETWEEN HYPONATREMIA,DIABETES,SMOKING AND HYPERTENSION.

Among patients with normal sodium levels 31% were diabetic, 40% were smokers and 40% were hypertensive.In patients who presented with hyponatremia on admission 67% were diabetic,50% were smokers,67% were hypertensive.In patients who developed hyponatremia within 72 hours after admission 33% were diabetic, 33% were smokers and 44% were hypertensive. Thus hyponatremia was more common among smokers,diabetic and hypertensive individuals.This is in accordance to the studies conducted by Goldberg A , Hammerman H et al,Aziz M et al,Hilis et al[71].

ASSOCIATION BETWEEN HYPONATREMIA AND DYSLIPIDEMIA

54% of individuals had serum total cholesterol >200mg/dl.In hyponatremia group 8 patients had total cholesterol >200 mg/dl and 5 patients had total cholesterol <200mg/dl..

7 patients with normal sodium levels,2 patients with hyponatremia on admission and 5 patients with hyponatremia within 72 hours had LDL levels>130mg/dl. Simillar results were found in Goldberg A , Hammerman H et al,Aziz M et al “s study.

ASSOCIATION BETWEEN HYPONATREMIA AND ANTERIOR WALL INFARCTION

The incidence of anterior wall MI among patients with normal sodium levels,hyponatremia on admission and patients who developed hyponatremia within 72 hours was 66%,67% and 89% respectively.This was higher than the results of golberg"s study which was 37%,49% and 45% respectively. Studies done by Krumholz[72] et al,Hillis et al also showed that hyponatremia was common in anterior wall infarction.

ASSOCIATION BETWEEN HYPONATREMIA AND KILLIP CLASS

66% of patients with hyponatremia on admission belonged to killip class I and 33 % belonged to killip class II.Among patients with hyponatremia within 72 hours ,77% belonged to killip class I, 11% belonged to class II and 11 % belonged to class III .Our results were similar to the studies done by Kerry Lee and Eric J Topol et al, Hillis et al and Goldberg A et el.

ASSOCIATION BETWEEN HYPONATREMIA AND EJECTION FRACTION

The mean ejection fraction was lower among patients who presented with hyponatremia(mean EF 43.33%) or developed hyponatremia within 72 hours(mean EF 47.33%) when compared to patients with normal sodium levels(mean EF 54.4%). Our results were consistant with the study conducted by Goldberg A ,Hammerman H et al, were the mean EF among patients with normal sodium levels,hyponatremia on admission and hyponatremia within 72 hours was 47%,42% and 42% respectively.

ASSOCIATION BETWEEN HYPONATREMIA AND MORTALITY

The overall mortality rate in our study was 22%.Mortality among patients with normal sodium levels was 11 %,mortality with hyponatremia on admission was 83%, and mortality with hyponatremia within 72 hours was 22 % .

In study done by Goldberg et al, the overall mortality rate was 10% . Mortality among patients with normal sodium levels was 6.2%.With hyponatremia on admission mortality was 20 %, with hyponatremia within 72 hours mortality was 17 % . In comparision with the above study, our study had higher mortality in patients with hyponatremia on admission where as mortality was almost equal in patients who developed hyponatremia after admission.

ASSOCIATION BETWEEN SEVERITY OF HYPONATREMIA AND MORTALITY

4 patients had a sodium level of <130meq/l and the mortality rate was 100%. Among patients with sodium levels between 131-134meq/l,mortality was 27% (3/11 patients).In comparission with other studies by Goldber A, Aziz M et al and Rahman et al [73]our study had a higher mortality rate.

From our study it is evident that patients who presented with hyponatremia or developed hyponatremia early after admission were males belonging to a higher age group,had diabetes,hypertension,smoking history ,anterior infarction and lower ejection fraction. This is in accordance to the studies conducted by Goldberg A,Hammerman H et al,Aziz M et al.

It was seen that serum sodium levels was statistically significant in determining mortality. Among survivors mean serum sodium level was 135.02±1.65 and lowest level was 133meq/l. Among non survivors mean serum sodium level was 130.9±2.7 and lowest level was 127meq/l.

Multivariate analysis showed that hyponatraemia on admission or early development of hyponatremia in patients with acute ST-Elevation MI appeared to be a strong independent predictor of short term mortality.

Prognosis worsens with increasing severity of hyponatraemia. Plasma sodium levels may serve as a simple marker to identify patients at high risk.

CONCLUSION

CONCLUSION

1. Asians are prone to develop STEMI at a youger age than western population.

2. In Indians hypertension, smoking, dyslipidemia, diabetes are predominant risk factors for STEMI.

3. Hyponatremia on admission or early development of hyponatremia within 72 hours was associated with a poor prognostic outcome.

4. Severity of hyponatremia predicts mortality.

Sodium levels <130 meq/l was associated with 100% mortality when compared to levels between 131-134 meq/l which had a mortality rate of 27%.

5. Patients with hyponatremia on admission or those who developed hyponatremia within 72 hours were males belonging to a higher age group, with lower ejection fraction, anterior wall infarction and a higher proportion of them were smokers,hypertensive,diabetic and had dyslipidemia.

6. Along with other risk factors, hyponatremia on admission or early development of hyponatremia appeared to be a significant independant risk factor in predicting short term mortality in acute myocardial infarction.

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PROFORMA

HYPONATREMIA - A PREDICTOR OF SHORT TERM MORTALITY IN ACUTE ST- ELEVATION MYOCARDIAL INFARCTION

PROFORMA

NAME: AGE: IP NO: DOA DOD

ADDRESS:

PRESENTING COMPLAINTS:

A. CHEST PAIN:

B. BREATHLESSNESS:

C. COUGH

D. PALPATION

E. SYNCOPE

F. SWELLING OF LEGS/ FACE

G. NAUSEA /VOMITING

RISK FACTORS-DIABETES /HYPERTENSION / SMOKER

GENERAL PHYSICAL EXAMINATION

VITAL SIGNS Pulse Blood pressure

SYSTEMIC EXAMINATION

CVS - RS -

ABDOMEN - CNS-

KILLIP CLASS-

INVESTIGATIONS

1) TROPONIN T.

2)BIOCHEMISTRY

RBS mg/dl BLOOD UREA mg/dl S.CREATININE mg/dl

SERUM SODIUM LEVELS

ADMISSION	24HRS	48 HRS	72HRS

TOTAL CHOLESTEROL mg/dl

HDL CHOLESTEROL mg/dl

LDL CHOLESTEROL mg/dl

VLDL CHOLESTEROL mg/dl

TRIGLYCERIDE mg/dl

IV.ELECTROCARDIOGRAPHY

V.ECHOCARDIOGRAPHY- EJECTION FRACTION

DIAGNOSIS

IN HOSPITAL COMPLICATIONS

DISCHARGE STATUS

FOLLOW UP UPTO 30 DAYS

ABBREVIATIONS

LIST OF ABBREVIATIONS

AMI : Acute myocardial infarction

AVP : Arginine Vasopressin

CAD :Coronary Artery Disease

CCF : Congestive Cardiac Failure

CK-MB : Creatinine Kinase-MB

CVD :Cardiovascular diseases

ECG : Electrocardiogram

IHD :Ischemic Heart Disease

LVF : Left ventricular Failure

MR :Mitral Regurgitation

TR :Tricuspid Regurgitation

DM :Diabetes mellitus

AWMI :Anterior wall myocardial infarction

ASMI :Antero septal myocardial infarction

IWMI :Inferior wall myocardial infarction

RVMI :Right ventricular myocardial infarction

MASTER CHART

S.NO	NAME	AGE	SEX	IP NO	DM	HT	SMOKING	PRIOR DIURETIC THERAPY	PRIOR CAD	EF	SODIUM ON ADMISSION	SODIUM AT 24 HRS	SODIUM AT 48 HRS	SODIUM AT 72 HRS	SBP	DBP	RBS	BLOOD UREA	S.CREATININE	S.CHOLESTROL	LDL	KILLIP CLASS	DIAGNOSIS	IN HOSPITAL COMPLICATIONS	OUTCOME	FOLLOW UP PERIOD
1	RAMAN	46	M	1106537	N	N	N	N	N	56	141	141	140	141	150	90	96	24	0.9	178	95	2	AWMI	2*HB	A	
2	RAVI	43	M	1106476	N	N	N	N	N	61	140	140	139	139	136	72	102	28	0.8	190	102	1	IPWMI		A	
3	JAYAKUM	53	M	1101866	Y	Y	N	N	N	66	139	137	137	138	150	100	118	30	1	224	121	1	IWMI		A	
4	PETCHIUT	60	M	1101698	N	Y	N	N	N	46	138	138	137	139	140	90	137	29	0.9	267	120	2	AWMI		A	
5	MUTHUKA	50	M	1102302	N	N	N	N	N	58	139	138	140	139	100	72	166	28	0.8	186	80	1	AWMI		A	
6	ELANGOV	60	M	1061420	N	Y	N	N	N	42	137	136	137	138	160	100	87	24	0.7	210	100	1	AWMI		A	
7	SENTHILKI	36	M	1047146	Y	N	Y	N	N	67	137	138	138	137	136	80	189	34	1.2	225	134	2	AWMI		A	
8	RAMADAS	68	M	1078786	N	N	N	N	N	56	133	134	134	134	100	70	112	31	1	188	108	3	AWMI	CCF	A	
9	PANJACH/	64	M	1077103	N	Y	N	N	N	47	137	134	134	132	180	110	121	24	1	256	178	1	IWMI		A	
10	SHANKAR	40	M	1077000	N	N	N	N	N	67	138	135	137	138	124	70	109	37	1.4	192	98	1	ASMI		A	
11	NALLAIYA	62	M	1076625	Y	Y	N	N	N	39	137	133	133	133	160	100	204	28	0.8	250	144	1	IWMI		A	
12	JEEVANA	58	M	1077934	N	N	N	N	N	55	137	138	138	137	130	80	78	38	1.6	214	120	1	IPWMI		A	
13	DHANAPA	49	M	1078088	Y	N	Y	N	N	41	128	129	130	129	126	72	156	34	1.2	184	92	2	AWMI	ACUTE MR	D	
14	KALIYAPEI	50	M	1078984	Y	Y	Y	N	N	36	129	130	130	129	170	110	216	21	0.9	255	164	1	IPWMI		D	
15	GUNASEEI	58	M	1079500	Y	N	Y	N	N	69	136	137	141	139	112	80	125	29	0.9	178	95	1	AWMI		A	
16	RAJKANA	32	M	1109871	N	N	N	N	N	72	138	138	137	138	130	80	136	28	0.8	232	126	1	ALMI		A	
17	JAYSHANI	32	M	1080069	N	N	N	N	N	51	137	141	138	137	110	80	108	24	0.7	174	96	1	AWMI		A	
18	DEVAN DO	39	M	1080653	N	N	N	N	N	39	140	137	137	138	130	90	128	34	1.2	166	86	2	IWMI		A	
19	BOOMINA	47	M	1080575	Y	Y	Y	N	N	43	128	130	129	128	150	90	178	22	0.6	189	91	1	IWMI		D	
20	MATHIMA	32	F	1081069	N	N	N	N	N	62	138	135	135	136	120	70	118	27	1	256	142	1	AWMI		A	
21	JOHN	50	M	1081601	N	Y	N	N	N	31	136	137	141	139	140	90	156	31	1	210	126	2	AWMI	PE	A	
22	KAMU	67	F	1081833	Y	Y	N	N	N	49	137	135	136	137	90	60	312	24	1	194	102	1	AWMI		A	
23	VARADAR	52	M	1082084	Y	Y	Y	N	N	57	139	138	138	137	200	110	279	32	1.1	203	112	1	AWMI		A	
24	NAMACHI	52	M	1082123	N	N	N	N	N	68	136	134	133	134	112	76	90	30	1	145	89	1	IWMI	VT	A	pif
25	AMMAPO	63	F	1081168	N	N	N	N	N	61	136	137	141	139	110	80	87	29	0.9	253	146	1	ALMI	2*HB	A	
26	GOVINDA	68	M	1083137	N	Y	Y	N	N	56	137	138	138	137	160	100	114	28	0.8	224	114	2	IWMI	PE	D	
27	SUNDARA	65	F	1083386	Y	N	N	N	N	48	141	142	141	141	110	80	224	24	0.7	151	94	2	AWMI		D	PIF
28	SEKAR	30	M	1083397	N	N	N	N	N	47	136	132	133	133	110	80	107	38	1.6	225	140	1	AWMI		A	
29	NAGENDR	41	M	1041359	N	Y	Y	N	N	68	139	139	137	138	160	100	120	34	1.2	192	112	1	IRWMI		A	
30	THENNAN	60	F	1041489	N	N	N	N	N	37	140	140	139	139	120	70	106	35	0.9	198	110	1	AWMI		A	
31	NILSON R/	45	M	1041870	Y	N	Y	N	N	46	136	134	133	134	120	70	267	29	0.9	224	112	1	AWMI		A	
32	BALA SAR/	34	F	1041954	N	N	N	N	N	59	137	133	131	133	110	80	77	28	0.8	215	107	1	AWMI		A	
33	AMUDHA	45	F	1042057	N	N	N	N	N	61	136	133	133	134	110	80	99	24	0.7	182	88	1	ASMI		A	
34	VELUSAM	80	M	1043460	N	N	Y	N	N	55	139	141	142	143	120	70	113	34	1.2	227	128	2	AWMI		D	
35	SENTHILKI	29	M	1043529	N	N	Y	N	N	35	137	141	138	137	112	76	86	38	1.6	236	138	1	AWMI		A	
36	GEETHA	60	F	1043828	N	N	N	N	N	52	137	138	138	137	112	76	90	34	1.2	171	95	1	IWMI		A	
37	ILAMPARU	39	M	1044596	N	N	Y	N	N	59	140	140	139	139	112	76	109	35	0.9	210	114	1	IPWMI		A	
38	STEPHEN I	47	M	1044607	N	Y	Y	N	N	71	138	138	137	138	160	100	256	34	1.2	195	98	1	AWMI		A	
39	KANNAN	37	M	1045032	N	N	Y	N	N	63	137	141	138	137	130	80	127	31	1	176	102	1	AWMI		A	
40	KANNAIYA	40	M	1043238	Y	Y	N	N	N	42	140	137	137	138	130	80	142	24	1	242	138	1	ASMI		A	
41	ANBALAG	68	M	1045422	N	Y	Y	N	N	48	138	141	140	141	170	110	116	37	1.4	244	141	1	AWMI		D	
42	RAVI CHA	45	M	1046425	N	Y	Y	N	N	50	137	141	138	137	130	80	105	28	0.8	205	102	1	AWMI		A	
43	EMMARAS	70	M	1046540	Y	Y	Y	N	N	30	137	134	132	132	80	60	112	34	1.2	190	105	3	AWMI	CCF	D	
44	MARUTHA	76	M	1047055	Y	N	Y	N	N	29	137	133	131	131	110	80	117	38	1.6	250	144	1	ASMI		D	PIF
45	KARRUPU	58	M	1047154	N	N	Y	N	N	43	140	140	139	139	130	80	98	34	1.2	192	98	1	AWMI		A	
46	ANNAMA	56	M	1096709	Y	Y	N	N	N	57	136	137	141	139	130	80	218	35	0.9	228	136	1	IPRVMI		A	
47	SUMATHI	38	F	1095661	N	Y	N	N	N	36	133	132	132	134	130	80	131	31	1	214	120	1	AWMI		D	
48	MURUGAI	62	M	1096328	Y	N	N	N	N	67	137	141	138	137	170	110	189	24	1	180	98	1	IPWMI		A	
49	NAGAMM	55	F	1096497	Y	Y	N	N	N	48	127	129	129	130	170	110	146	34	1.2	243	154	1	AWMI		D	PIF
50	MOHAN	62	M	1078418	Y	Y	Y	N	N	44	140	140	139	139	190	100	98	35	0.9	226	110	1	AWMI		A	

KEY TO MASTER CHART

DM :Diabetes mellitus

HT : Hypertension

CAD : Coronary artery disease

EF : Ejection fraction

SBP : Systolic blood pressure

DBP : Diastolic blood pressure

RBS : Random blood sugar

2*HB : Second degree heart block

CCF :Congestive cardiac failure

PE :Pulmonary edema

VT : Ventricular tachycardia

PIF :Post infarction Angina

A :Alive

D : Death

AWMI : Anterior wall myocardial infarction

ASMI : Antero septal myocardial infarction

IWMI : Inferior wall myocardial infarction

RVMI : Right ventricular myocardial infarction